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U.S. DEPARTMENT OF COMMERCE
PATENT AND TRADEMARK OFFICE

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Serial No.

09/688,015

Applicant

Junying Yuan et al.

Filing Date

October 13, 2000

Group

1614

IDS Filed

March 9, 2001

Customer No.

21559

INFORMATION DISCLOSURE
STATEMENT BY APPLICANT
(Use several sheets if necessary)


(37 CFR §1.98(b))

U.S. PATENTS

Examiner's Initials	Patent Number	Issue Date	Patentee	Class	Subclass	Filing Date (If Appropriate)

FOREIGN PATENT OR PUBLISHED FOREIGN PATENT APPLICATION

Examiner's Initials	Document Number	Publication Date	Country or Patent Office	Class	Subclass	Translation (Yes/No)

OTHER DOCUMENTS (INCLUDING AUTHOR, TITLE, DATE, PLACE OF PUBLICATION)

AMD	Borner et al., "Apoptosis without caspases: an inefficient molecular guillotine?," <i>Cell Death Differ.</i> 6:497-507 (1999).
AMD	Büyükbıngöl et al., "Studies on the synthesis and structure-activity relationships of 5-(3'-indolal)-2-thiohydantoin derivatives as aldose reductase enzyme inhibitors," <i>Il Farmaco</i> 49:443-447 (1994).
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AMD	Fiers et al., "More than one way to die: apoptosis, necrosis and reactive oxygen damage," <i>Oncogene</i> 18:7719-7730 (1999).
AMD	Herceg et al., "Failure of poly(ADP-ribose) polymerase cleavage by caspases leads to induction of necrosis and enhanced apoptosis," <i>Mol. Cell Biol.</i> 19:5124-5133 (1999).
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AMD	Holler et al., "Fas triggers an alternative, caspase-8-independent cell death pathway using the kinase RIP as effector molecule," <i>Nature Immunol.</i> 1:489-495 (2000).
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AMD	Kitanaka et al., "Caspase-independent programmed cell death with necrotic morphology," <i>Cell Death Differ.</i> 6:508-515 (1999).


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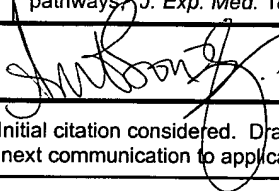
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AMD	Leist et al., "Inhibition of mitochondrial ATP generation by nitric oxide switches apoptosis to necrosis," <i>Exp. Cell Res.</i> 249:396-403 (1999).
AMD	Li et al., "Induction of necrotic-like cell death by tumor necrosis factor alpha and caspase inhibitors: Novel mechanism for killing virus-infected cells," <i>J. Virol.</i> 74:7470-7477 (2000).
AMD	Lüschen et al., "Sensitization to death receptor cytotoxicity by inhibition of fas-associated death domain protein (FADD)/caspase signaling. Requirement of cell cycle progression," <i>J. Biol. Chem.</i> 275:24670-24678 (2000).
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